

Bacterial Skin Infection

2

16/5/2015

1. NL Skin Flora:

Class	Organism	Location on Body
Aerobic Cocci (+ve cocci)	<ul style="list-style-type: none"> Staph <u>Epidermidis</u> " <u>Hominis</u> " <u>Luteus</u>, <u>Micrococcus</u> 	All body sites Specially <u>intertriginous</u>
Aerobic - Coryneform (+ve rods)	<ul style="list-style-type: none"> <u>Corynebacterium minutissimum</u> (erythrasma) عراج " <u>Xerosis</u> 	<u>Intertriginous</u>
Anaerobic Coryneform (-ve rod-shaped)	<ul style="list-style-type: none"> <u>Propionibacterium</u> <ul style="list-style-type: none"> <u>Acnes</u> <u>Avidum</u> <u>granulosum</u> 	on skin especially Sebaceous glands & follicles.
G - ve	<u>Acetobacter</u> spp	<u>Intertriginous</u>
Yeast	<ul style="list-style-type: none"> <u>Pityrosporum</u> <u>ovale</u> <u>orbiculare</u> (<u>Malassezia</u>) (normal commensal yeast (skin flora)) (lipophilic) 	skin rich in sebaceous glands (scalp, upper chest back)

NB Staph aureus: Not member of Resident flora but it may be found in:

- Ant. Nares → 30%
- Perineum → 20%
- AXillae & Toe Web → 10%
- Atopic skin → 90%

off population

recurrent abscesses with no wound
Folliculitis (infection)
Abscess

NB Colonization

Staph aureus
commensal on skin

② Bacterial skin infection includes:

(2)

exacerbate ATOPY.

exacerbate Psoriasis

① Gram +ve:

Staph & Strep
epi & e

- Staph. aureus
- Strep. pyogenes
- Corynebacteria v
- Others:

... clostridial inf.

- Bacillus anthracis
- Bacillus cereus

• Corynebacterium diphtheriae

①: Erysipelothrix rhusiopathiae → Erysipeloid

② G-ve:

- Pseudomonas Aeruginosa
- Neisseria Meningitidis

- Bartonella
- Brucella
- Burkholderia

cat scratch dis
Angiomatosis
Bartonellosis.

①

• MalaKopla Kia →

② (HL)

- Salmonella
- Klebsiella
- Francisella

• Yersinia pestis

• Haemophilus influenzae

- Streptobacillus moniliformis
- Anaplasmosis & Ehrlichiosis

③ Spirochaetes: → T. pallidum

✓ Borrelia Burgdorferi

②

• Endemic (non Venereal) Trypanomastix

Yaws

Pinta

③

④ Bacteria previously classified as Fungi:

✓ Actinomyces

✓ Nocardiosis → Actinomyces

CIP

• Mycetozoa

It is not a fungus

Ex: (1) e. prini

Diseases Caused by Staph aureus

(3)

1- Direct infection: (Tend to invade appendages):

- Impetigo (Non bullous) (by swap \rightarrow Staph aureus)
- Ecthyma (\pm)
- Superficial folliculitis \leftarrow Jais HF \rightarrow Staph
- Syccosis Vulgaris
- Furuncle (Boil)
- Carbuncle
- Abscess
- Pyomyositis. (بوليا)

2- 2ry infection: of ulcers Burns Eczema

3- Toxins \leftarrow mediated \leftarrow by swap \rightarrow (no staph present)

. Bullous impetigo

. SSSS

. TSS \leftarrow (Toxic Shock synd)

. Staph. scarlatina

Granuloma 4- Botryomycosis (granulomatous staph. inf.)

5- Pyoderma Vegetans.

6- dis. influenced or

provoked by it e.g. AD \checkmark (A Topic Dermatitis)

Impetigo

(4)

Def Staph &/or strept inf. of superficial Epid.

Aetiology:

• Bullous Impetigo → staph. (it represent localized form of SSSS)

• Non Bullous → staph & strept. (staph > strept)
(Imp. Contagiosa)

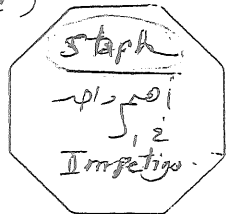
NB the 1st pathogen in both types is staph. & less commonly strept.

Recently 2005: (in Non Bullous): staph

• developed countries → most prevalent in both types

• developing → prevalence of S. Pyogenes.

most infect- start as strept → Later replaced by staph. (staph. & produce toxins that kill strept)



So explain why staph always isolated

Non Bullous (Crusted) (Contagiosa)

Bullous Impetigo (Circinate)

• more common (For 7 cases)

• less common

• Children.

• Neonatal

Any site : Common : Face (perioral, perinasal)
Limbs, scalp (Except Palms & Soles)

Any site : Common : Face
Including : Palms & Soles (J.L)

Erythem. macule → Vesicle → Pustules → Rupture → honey-colored yellow Crust (Crusted Impetigo)

Small Vesicle → Superficial Flaccid Bullae (Circinate)
Collarette scales (But No Crust & No surrounding Eryth.)

Mild L.N & systemic manif.

• No L-N or systemic manif. but ± Weakness, diarrhoea & Fever

1- Subcorneal blisters

1- Sub. st. granulosum blister filled w Eos.

2- Few acantholytic cells

2- Acantholytic cells

3- st. malpighii → Spongiosis & Neut.

3- upper dermis (PMNL)

4- +ve Bact. in lesions. (staph & strept)

4- No Bact. in lesions

5- upper dermis : intense N.E

staph (impetigo)

[In SSSS → Free]

Complications

5

Non Bullous

Acute Post Streptococcal GN (APSG)

~5% of cases of non bullous impetigo caused by Strept. pyogenes (GABHS)
Serotypes: 1, 4, 12, 25 & 49

after Latent period (3 wks)
(in Sore throat Latency is 10 ds)

Risk: is not altered by antibiotic
[child → 6y]

Bullous

In Immuno Compromised Patients: it ± → SSSS.

Usually Age < 6y
Prognosis:

Excellent: in children
Bad: in Adults.

NB: Impetigo don't cause Rheumatic Fever

DD:

Bullous

- Bullous papular urticaria
- HSV
- Thermal burns
- Bullous diseases.

Non Bullous

- Insect bites
- Eczema
- HSV
- Varicella
- Candida.

Treatment:

- Crust Removing BY Wet dressing application
H₂O₂ or olive oil
Ed + J + CBU
net 1: ✓
- Antibiotic:

Topical

Systemic

Fucidin

Mupirocin

Conc 1% / 2%

Indications:

- Healthy Patients
- Limited dis
- Superficial lesions
- No systemic mants

Indicated if: (see)

- Severe dis
- deep impetigo & immuno compromised
- systemic mants or L.N.
- scalp impetigo

Some NBs on Impetigo:

- Mild dis. in healthy patient with out Systemic manif. → Treat @ Topical only (Equal or better than Systemic)

2 Types of Impetigo: 6

- Bullous
- Non Bullous (Imp. Contagiosa)
- Circinate
- Crusted
- Common: When infect. occurs in preexisting wounds
- Ecthyma

3 lines of Ht:

(NB)

1. Cefixim tab	Cefuroxime
2. Cefixim	3. Zinnat
- No tab	125 (sus)
- Susp. & Vial	250
	500
	250
	750
	500

Uncomplicated

- mild dis.
- Healthy pt.
- No Systemic manif.

1st line

Topicals

Fucidin
Mupirocin

2nd line

Oral

- Penicillins (Beta lactamase resistant)
- Cephalosporins (1st or 2nd Gen.)
- Macrolide

Complicated IV

1st line

(3rd Gen) Ceftriaxone (IV)

Rocephin 500
Ceftriaxone

2nd line

- (2nd Gen.)
- Cefuroxime
- Ampicillin
- Sulbactam
- (Unasyn) 3.75g tab

Uniclam
Vial 750
1500
Tab: 375

When Dealing @ impetigo always put in your mind MRSA..!!

NB: Clinical Tricks: Ht of impetigo

1 Soak Crusted areas

گازان ماء + جل توضع لى 10 دقايقه
كذا مرة يوميا
Remove Crust

2 Fucidin

3 Systemic

Ecthyma

involves epidermis & upper dermis

(7)

Def → deep ulcerating form of non Bullous impetigo; That's caused by the same pathogen of impetigo but infection & inflammation extend to the upper dermis → Thick crust & ulcer → Scarring. (whole Epid + upper dermis).

organism: (Strept) Common > Staph

CIP: initial Vesicle or Vesiculo pustules → Enlarge → Hgic crust separate → punched out ulcer & Purulent necrotic base → slow healing → Scarring.
Site: Commonest at shin & dorsum of feet.

Risk factors:

1. Young age (children)
2. old age
3. Immuno suppression
4. poor hygiene.
5. Neglected impetigo
6. minor Trauma (Insect, scratching & dermatitis)
7. High Humidity & temp.

(NB Staph)
initiate the lesion or 2nd inf pre existing around.

Complications:

- Staph. Contaminant.
- Bacteremia.
- Cellulitis
- Osteomyelitis.

DD

→ Ecthyma gangrenosum
→ Vasculitic ulcers.

Treatment

Systemic Rx: as impetigo

Wash & Soap then use → Fucidin or Mupirocin

Improve Hygiene:

- ① غسل يدينا بالماء والصابون.
- ② تغيير الملابس والملابس.
- ③ استخدام ملابس نظيفة.
- ④ Avoidance of Trauma.

4. Staph inf. related to Hair follicles

(8)

1. superficial pustular Folliculitis (Follicular impetigo of Boekhart).
infect- of Follicular
Ostia ← opening
2. SYCOsis Vulgaris = (SYCOsis barbae) : infection of (whole)
(depth of Follicle)
(of Beard & Moustach.)
3. Furuncles = Boils:
Staph. inf. of whole (depth of the Follicles)
+ (Surrounding dermis.)
4. Carbuncle: Contiguous collection of inflamed
Follicles (grouping of ≥ 2 Furuncles)
inf. extend to deep S.C.T → more
Severe Sympt.

NB ① Abscess: Can occur any where (but) Furuncle
affect the hair follicles.

② All Furuncles are d.t Staph; (but) anogenital recurrent
Furuncle, G-ve may be involved.

CIP

1. Boekhart impetigo: dome shaped pustule at the
Orifice of the follicle.
2. SYCOsis barbae: Follicular Papules & pustules (that)
may remain discrete or coalesce into plaques.
← Affect beard & Moustach of adult males.

↑ NB: Lupoid SYCOsis: Follicles destroyed by
scarring & pustules fringe the advancing
margin around pink atrophic scar.

lupus
like.

3- Furunculosis : Predisposing factors :

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1. ↓ Immunity e.g DM & HIV
2. Itchy Skin e.g scabies & miliaria
3. Alcoholism
4. Obesity

Tender red Nodule → Enlarges → discharge
pus → scarring ← deeper deeper

4. Carbuncle: swollen, painful, suppurating area (plaque)
discharging pus from several openings
or sinuses; Commonest on Nape of diabetics

NB For furuncle & carbuncle at first → Drainage → then, Antibiotics

Any site can be affected by furuncle & Carbuncle but the commonest are:

- ↳ Face & Nape of Neck
- ↳ Chest
- ↳ back
- ↳ axillae &
- ↳ Buttocks.

أدور على

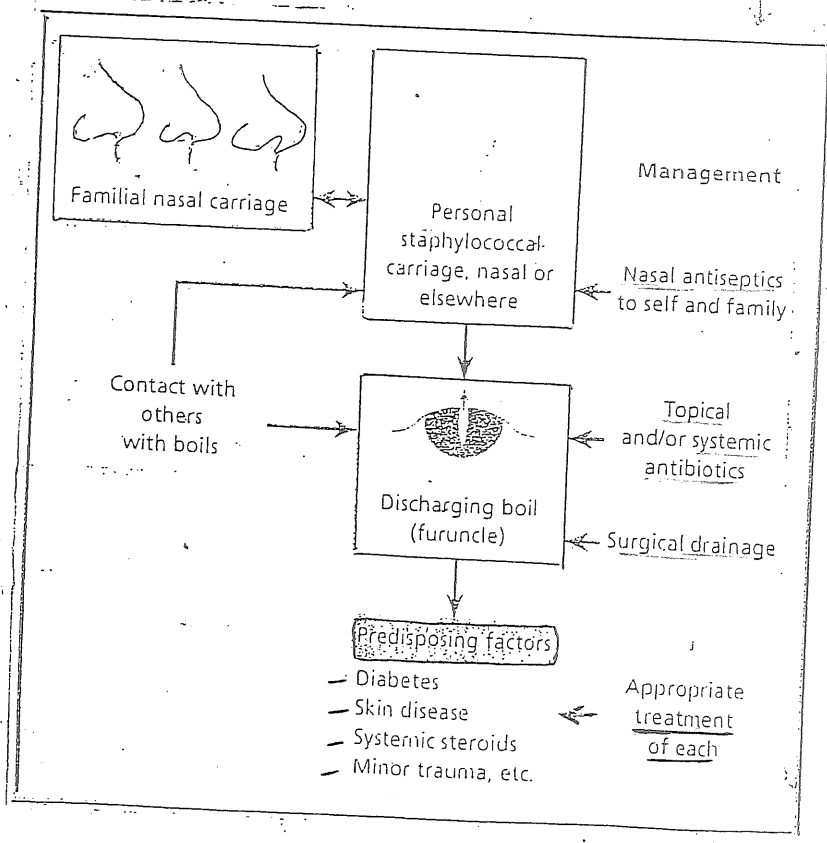
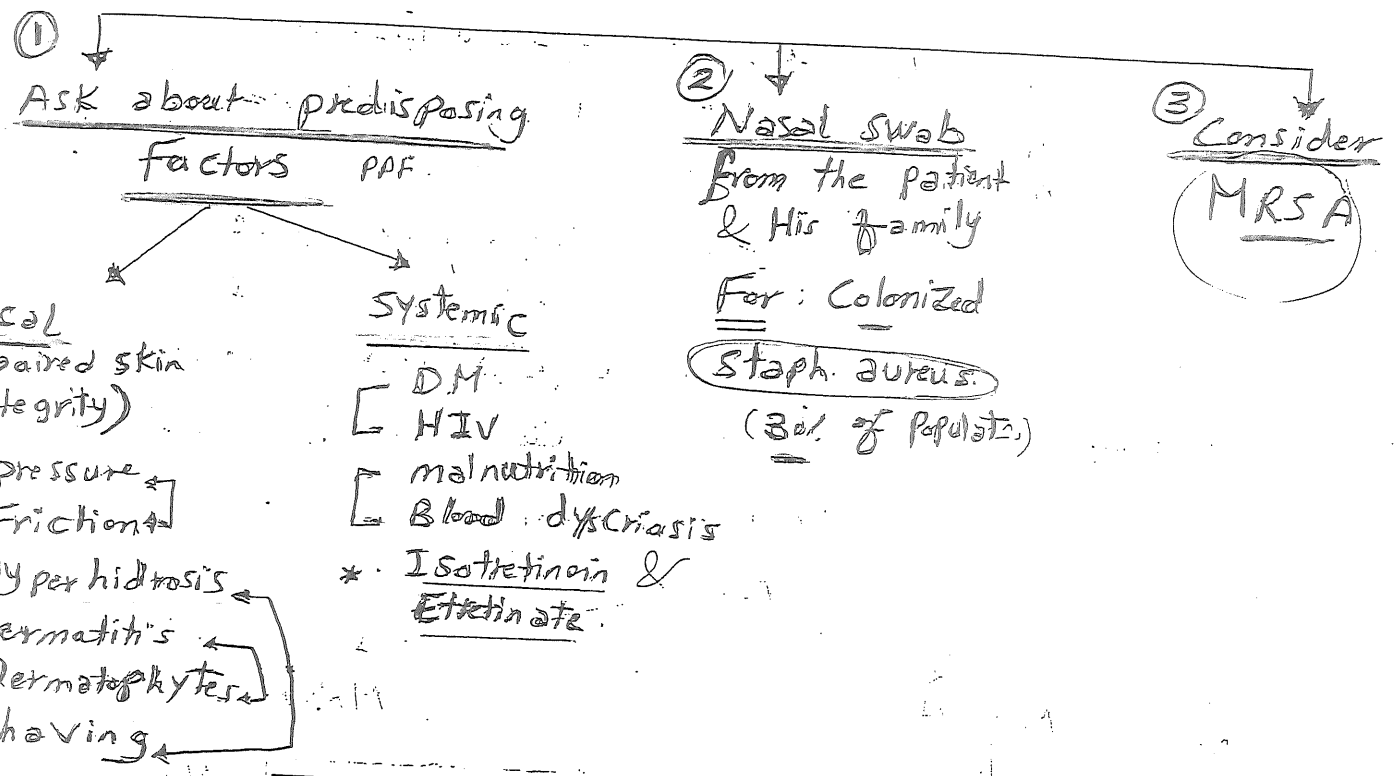
chr. or Recurrent Furunculosis

(حرفين بيحي يد عامل وخارج مسمرة)

أدور على

قاعة

When you see this Case Put in Your mind 3 points.



② Nasal swab

③ MRSA and etc.

① PPF

Treatment

(A)
(B)

A DeColonization measures

(For: Nose, axillae, Perineum)

1. يتم استعمال مطهر الاستياقلون يوميا لكل الجسم او Bleach.

2. غسل الأيدي باستمرار. (استعمال بكتون أو (ستياقلون) (axillae, groin, Hands) Bath.

3. غسل الملابس والمفارش باستمرار. (as in AD)

4. دهان "فينوسيدين" مرتين يوميا حول فتحة

الأنف والشرج [or Mupirocin] (Flexures) "الأنف" وكذلك في

B Systemic Antibiotics For Eradication of Staph.

For

MSSA

Rifampacin (2 Caps/d) 300mg

+

Flumox (2 Caps/d)

↓

علاج 10 أيام

MRSA

(i). TMP + SMX

(ii). Minocycline or Doxy.

(iii). Clindamycin

كبسولة (150) يوميا 3 مرات

if failed

نظف ودرع على
قناة تصريف

IV Vancomycin

S.E: ① Pain

② thrombophlebitis

③ Red Man Synd:

سببه

Most cell
degranulat.

- Flushing & erythematous rash appearing 4-10 mins after start or completion of infusion.

④ LABD

مقاومة

Methicillin Resistant Staph aureus

"R" (12)

(MRSA)

Def → Strain of staph aureus that's resistant to Large Group of Beta-lactamase resistant antibiotics as penicillins & Cephalosporins.

Methicillin
Dicloxacillin
Nafcillin
Cloxacillin

AET. Presence of "mecA" gene in the bacteria → alteration of site at w Methicillin binds to kill the organism.

BP2a

Infection Caused by MRSA:

↓ — The same inf. as other staph because the organism itself is not any more virulent or infectious.

↓ MRSA can colonise body sites as usual

Staph eout causing "Sickness" (see staph aureus colonization)

So the problem with

MRSA is d.t

Antibiotic resistance

not virulent.

Types of MRSA

or Hospital Acquired

① Health care ass. MRSA (HA-MRSA) = Nosocomial Transmission

② Community ass. MRSA (CA-MRSA) = among population
(Less common)

is or
equivalent

Clinical presentation of CA-MRSA (As staph)

Furunculosis (most common) → Abscess or cellulitis
impetigo
SSSS
✓ Life threatening e.g. Bacteremia, Septic shock, TSS: } Less common

Diagnosis of MRSA:

(13)

at first (suspect) MRSA if:

- ① prevalence of MRSA in a given community
- ② possibility of Nosocomial inf.
- ③ Severe inf.

↓ do

Culture & sensitivity.

Treatment of MRSA:

① Decolonization Measures → see recurrent furunculosis.
 (also use of fluoroquinolones as they help colonization).

② Antibiotics:

septrin → like drug eruption.

- Sulfa
 - Tetracyclines
 - Clindamycin
- (CA-MRSA are sensitive but HA-MRSA are resistant)

HA-MRSA
 "مقاومة الجراثيم"

استعمل علاج ليو

مقاومة الجراثيم
 (مقاومة الجراثيم)

Vancomycin (CAMRSA ++ / HA-MRSA ±)
 Linezolid & Telapranin [but some strains are resistant]

- Daptomycin
 - Quino- & Dalfo Pristin
 - Tigecycline
- (مقاومة الجراثيم)

Empiric

NB, IV Vancomycin should be considered in:

- ① - Severe, Life threatening inf. in areas where MRSA is present
- ② - pts. with History of MRSA Colonization
- ③ - IV drug users.

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Beta-lactamase resistant Antibiotics

- either
- Methicillin, dicloxacillin, Cefoxacin, ...
 - Amoxicillin + B-lactamase inhibitor: clavulonic acid (E-mox-clav)
 - Ampicillin + " " : sulbactam (Uniclam or Unasyn)
- dose 50mg/kg/day

def → presence of inflammatory cells within walls & Ostia of Hair follicles → Follicular Based Pustules & Papules

NB: Perifolliculitis: presence of inflammatory cells in the perifollicular tissue & reticular dermis.

Predisposing factors

- frequent shaving
- Immunosuppression
- preexisting dermatoses
- Long term antibiotic use
- occlusive clothing or dressings
- Hot Humid conditions
- ✓ DM / obesity

Clinical presentation:

- Itchy (less commonly painful) follicular Pustules Pierced Centrally by hair.
- If No Pustules: The follicular Papules or Collarette of scales are clue for D.

3 Types of Folliculitis:

Not Itchy → 1- Superficial multiple small papules & pustules on an Erythematous base Pierced Centrally by hair (although hair may not be visualized)

Not Pain → 2- Deep Erythematous, often Fluctuating
→ (modules) → more Pain → Suppurative drainage & ± scarring & Hair loss.

③ Patterned folliculitis on areas of shaving or occlusion

Site: any hairy area can be affected but commonest.

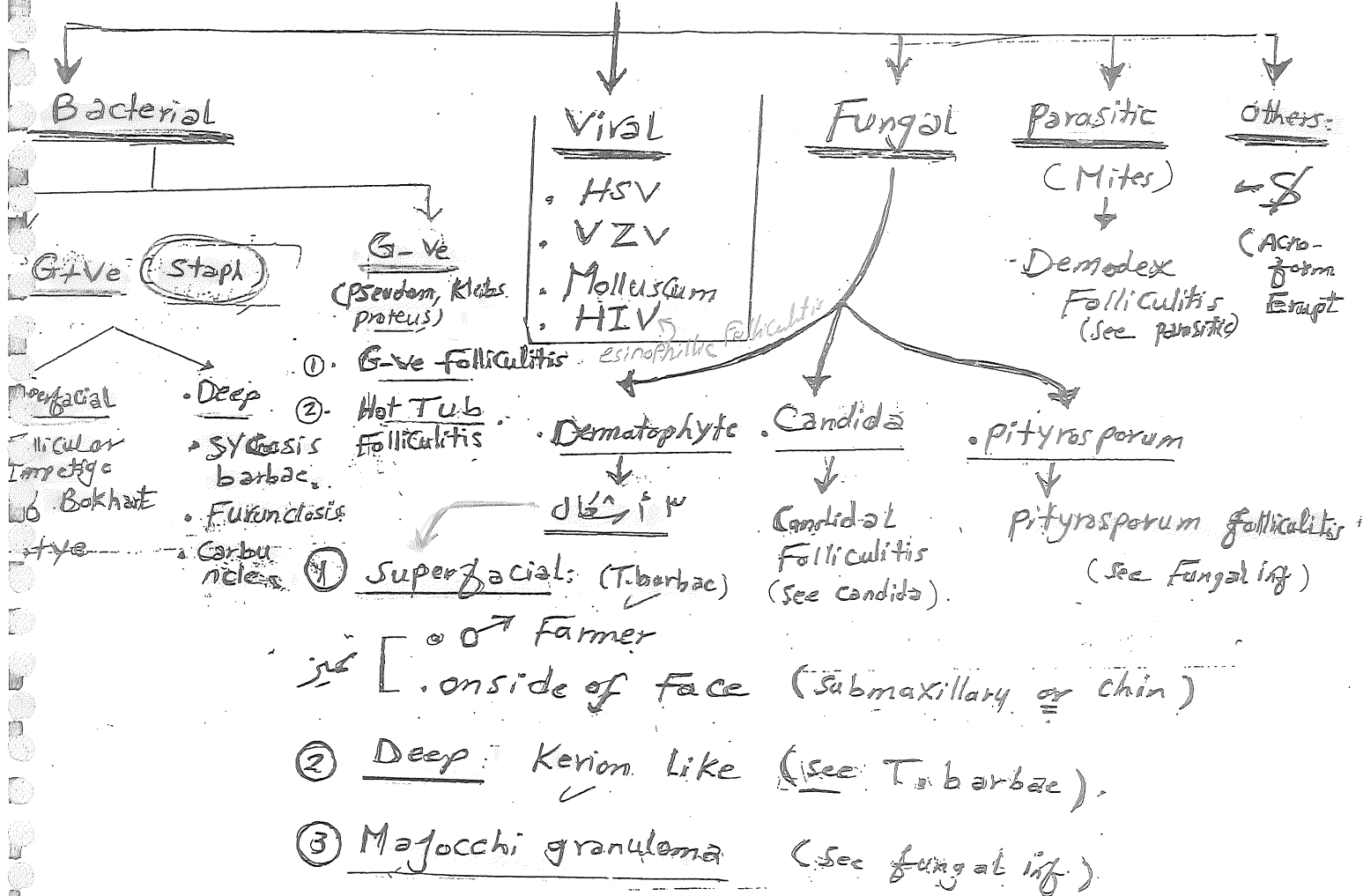
[Face
Scalp]

[Thighs
Axillae
Inguinal]

(usually on areas of occlusion &/or Terminal Hair)

Causes of Folliculitis ✓

① Infection:



② Irritation: إزالة أو شدة

- Waxing & Threading.
- Electrolysis
- Pseudofolliculitis barbae

③ Contact reaction:

- occlusion By
 - Moisturizers
 - Adhesive plasters

Topicals:

- ✓ Coal tar
- ✓ Cutting oils
- ✓ Cs (overuse & perioral dermatitis*)

Neutrophil
ass. 1ry
Scarring
Alopecia

④ Inflammatory skin dis.

(deep seated sterile
folliculitis that \rightarrow
scarring)

- F. decalvans
- Dissecting Cellulitis
- Erosive pustular dermatitis
- A. Keloidalis

- LP - Lichen planus
- DLE - Discoid LE
- Folliculitis decalvans

⑤ Immunosuppression

✓ Eosinophilic folliculitis (HIV ass.)
* severely itchy

↑ by HIV drugs \leftarrow Antiviral
Therapy of HIV (HAART)

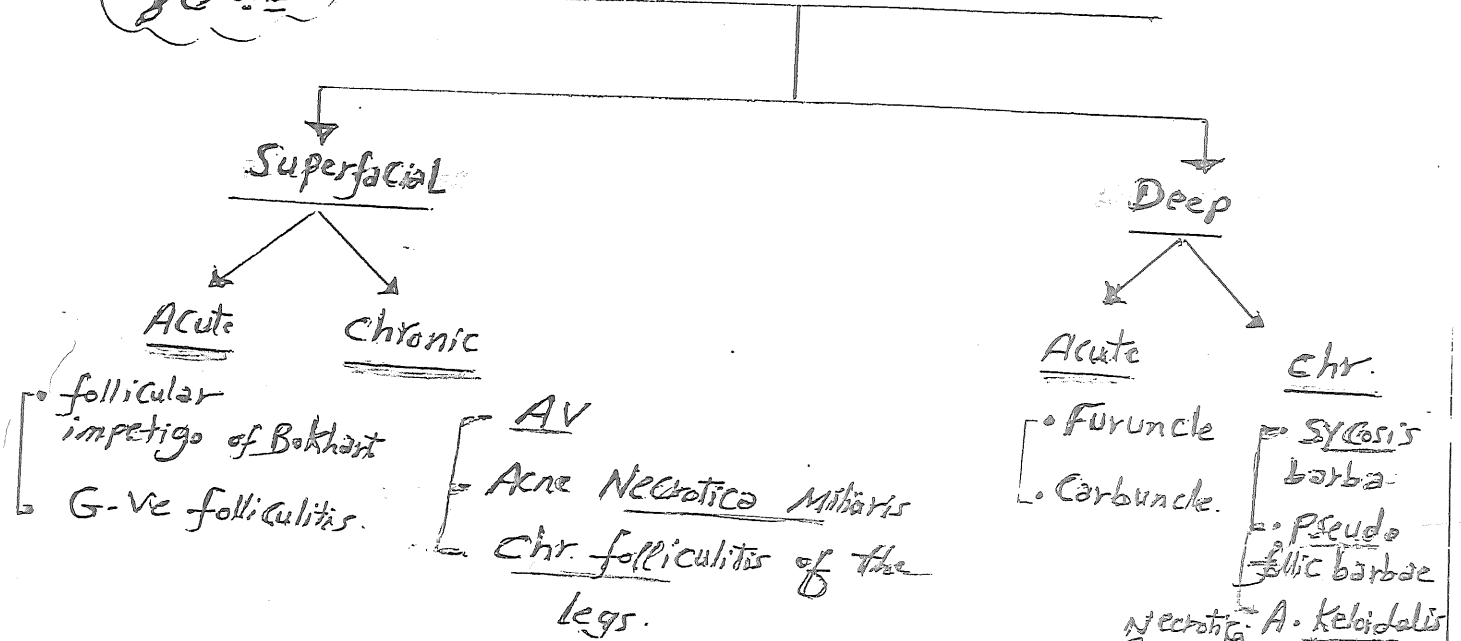
⑥ Acne / variants

- Acne Vulgaris
- Nodulocystic Acne
- Steroid Acne
- Rosacea
- Propionibact. folliculitis
- Acne Necrotica
- Acne Necrotica Miliaris
- Chloracne
- Acne Keloidalis Nuchae
- Hidradenitis suppurativa

⑦ Idiopathic

سليم في الجلد
طبيب في الجلد

Another Classification for folliculitis



3. Eosinophilic Folliculitis:

3 Varieties

سوال 19

(A) Ofuji dis: (Eosinophilic pustular folliculitis): -

• 30 Ys, ♂ Japanes (M:F = 5:1)

• discrete papules & pustules → Circinate arrangement

→ peripheral rim of pustules =

Central clearing at Acne prone sites.

(B) Immuno suppression Ass: (HIV ♂ Pt e CD4 < 300)

• Persistent

• No annular pattern

• Face, scalp & Trunk (Very itchy)

• FIARRT if failed → Antibiot, Antihistamines & UVB

(C) Infantile:

• 1st 24 hrs resolve First few wks.

• ♂ infant

• May be cyclic Course Lasts (ms - Ys)

• at scalp & Eye brow → ass e Crusting

• ± ass e peripheral Eosinophilia.

Invs for a case of Folliculitis:

(1) for Bact:

Gram stain

• detection of Nasal Carriers

(see recurrent furunculosis)

(2) for Fungal: KOH ex.

(3) Viral: Culture.

(4) Punch Biopsy: for other Cases e.g eosinophilic folliculitis & LP.

Treatment → according to the Cause.

1st line

Endometh-
acin

2nd

• UVB
• Dapsone
• Colchicine
• Cs

Trm ass

Leuk, Lymph

HIV
ass.

(#)

• Self limiting
• Symptomatic

Hydradenitis suppurativa

Hydradenitis suppurativa

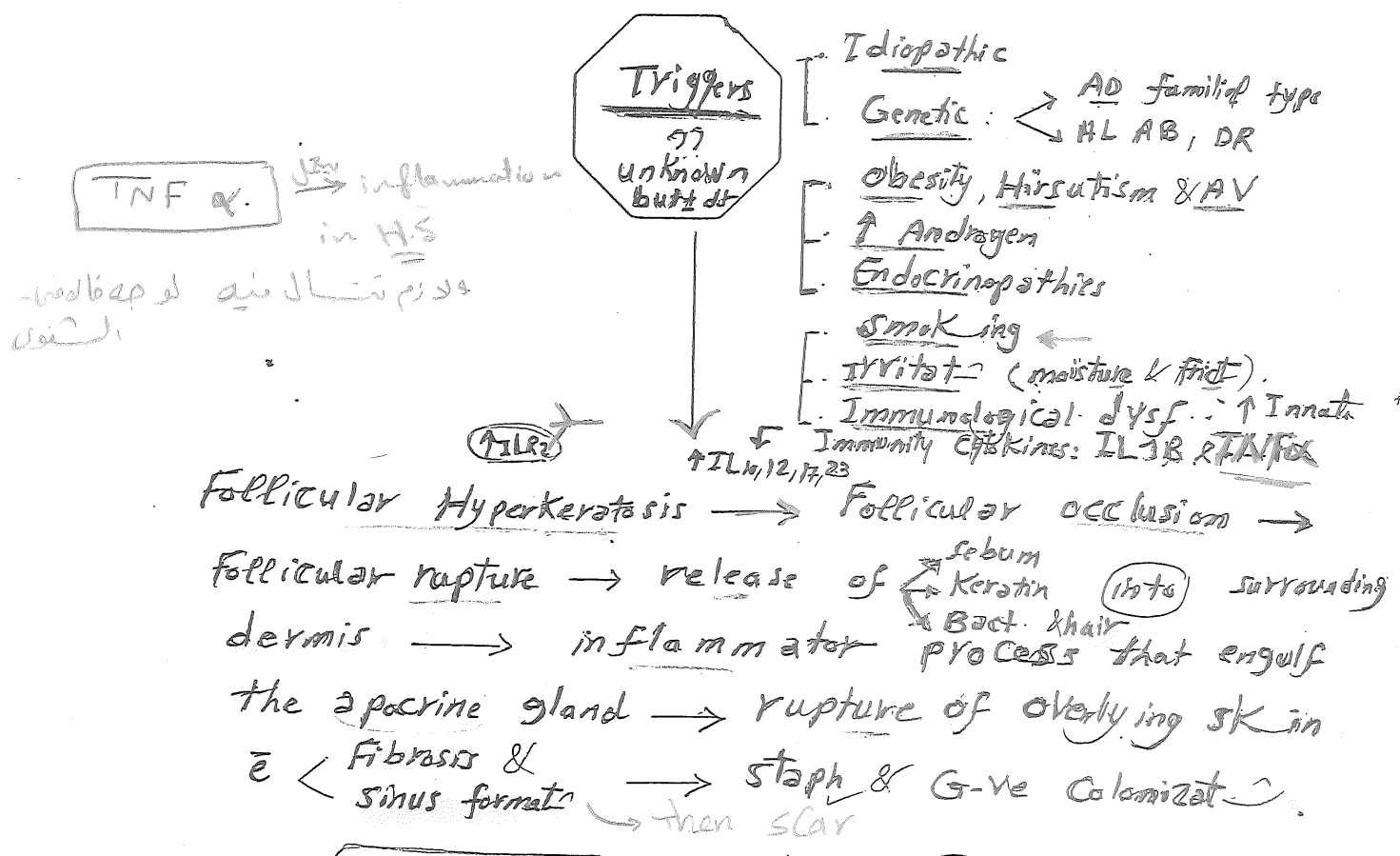
Hydradenitis
(برنيس)
DNNZ

Acne Inversa = Apocrinitis

Acne Inversa = Apocrinitis

Def Chr., recurrent inflammatory skin disorder
ch by recurrent abscess format - Primarily
in folded areas of skin that contains
both Terminal hairs & Apocrine glands.

Etiopathogenesis: follicular occlusion disorder ch by:



NB: 1ry apocrinitis is reported on 5% of cases so
It's Not 'apocrinitis' disorder but follicular epith disorder

Epidemiology:
• Age: Puberty & Post pubertal (11-50y)
• Sex: F > M (3:1) but [AV ≈ 23]
Perianal is M > F

Chronic recurrent Abscess like > 5 times/month (21)
CIP starts as small, red, Tender, S.C Nodule

→ suppurat → fluctuat → ^{Rupture} ^{through skin} scarring
 & format of sinus Tract → recurrence
 at ^{axillae} ^{groin} ^{intermammary} (NS Abscesses/m)

DNNZ → Distinct clinical staging have been defined

For the condition: (Hurley's Classification)

- Complicat:
- ↑ Metabolic Synd Incid.
 - Anemia
 - 2ry Amyloidosis
 - Lymphoedema
 - Fistula to ^{Urethra} ^{UB} ^{Rectum}
 - Contracture & limb limitat
 - Hypoproteinemia

NS - Arthropathy
 SCC

isolated = Stage I → Isolated abscess (solitary or Multiple) only
 recurrent = Stage II → Recurrent abscesses (" " ") widely separated + sinus tract format
 diffuse or broad = Stage III → diffuse or broad involvement & Multiple interconnected sinus tracts & abscesses

NB: Multiple Head ^{Black} Comedones also seen (Bihead & Trihead)

NePhrotic syndrome

- Pathology
- heavy mixed infilt. in lower dermis & S.C.T
 - sinus tracts & inflamm cells & ^{Keratin} debris
 - G.T & FB Giant Cells
 - in chr. cases → Extensive fibrosis + destruct of piloseb. follicles & sweat glands.

DD:

[1] Staph. Furunculosis & Abscess

pointed or ulcerate through the skin
 No sinus tract format
 not bilateral

GI

[2] ^{Cutaneous} Crohn's, Granuloma inguinale, Mycetoma & TB

[3] Elephantiasis Nostra Verrucosa: (2ry) to recurrent strept. Lymphangitis → may distort! ext. Genitalia.

1- General Measures:

- stop smoking → توقف التدخين
- ↓ W.t (if overweight) انقاص الوزن
- ↓ Friction & moisture (loose undergarments, absorbent Powder, antiseptics, Alum. chloride).

2- Topical tt:

- Clindamycin: chr. use (↓ staph carriage & 2ry inf.)
- ILCS: in early inflammatory lesions
- Others: Anti Septic Soaps. Mupirocin: to eradicate Staph Carriers.
- H₂O₂ ← Hydrogen Peroxide
- Alum. chloride ← عازل معدني

3- Systemic tt:

- ①. Systemic Antibiot
- ②. Retinoid
- ③. Antimal
- ④. Others
- Biologics

- CS: 60-80 ms (dramatic response that Flare on discontinuation)

- Cyproterone acetate (very large doses needed so ?? safety)
- CYP. + EE (Diane) → better

✓ Cyclosporine

✓ TNFα (Infliximab)

✓ Botox

Humira (Adalimumab)

FDA C. 12

Isotretinoin: (less effective)

Acitretin (Effective)

Finasteride (5 mg 1d for 3ms)

Systemic antibiotics → (according to Culture & Sensitivity) as anti-inflammatory

Others

4- Surgical tt: (Excision Not incision)

Surgical Excision of affected area then Closure

Surgical de-roofing (if not effective minimal invasion + office procedure tissue preservation)

CO₂ laser stripping + 2ry intention healing

(NB) Avoid incision & drainage → Scarring & sinus formation

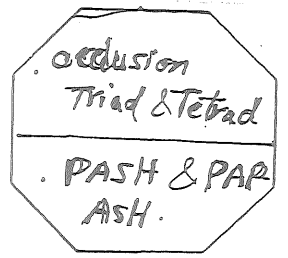
Amikacin
Infliximab
Etanercept

FDA C. 12 (Adalimumab)

N.B. low dose 10 mg 1 day
Acne
Doxyl + Flgyp

Synd with HS

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Idiopathic ①. Follicular occlusion Triad:

Follicular occlusion

on → axilla → ①. Hidradenitis suppurativa.

Sebaceous & Face ②. Acne Conglobata

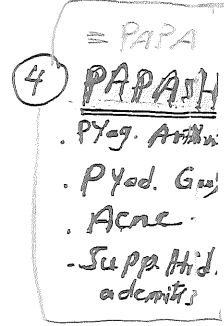
scalp ③. Dissecting Cellulitis of scalp.

② Follicular occlusion Tetrad.

③ PASH ^{PG} _{Acne} ^{HS}

• Follicular occlusion tetrad:

as triad + Pilonidal Sinus.



(Pilo = pilus = hair / Nidal = Nest)

(*) def. Chr. Condition Ch By Sinus Tract at perianal region (at base of spine bet clefts of buttocks)
• the cavity of 1 sinus is filled w Nests of Hair ± infected → Abscess.

• Sometimes a cyst called pilonidal cyst is present.

• Etiopathogenesis ?? may be dt:

1. Genetic → genetically prone to Follicular occlusion (Triad or Tetrad)

2. Some borne w small holes or pits at this site w is actually enlarged Hair follicle → Frict & mat → hair shaft Pokes through wall of follicle → inflammatory FB react.

* 3. Neighbouring Hairs or free hairs from other parts of body collect in 1 Pit & invade the small opening created by 1 distorted follicle.

4. Skin & perineal bact are superadded.

• Risky .

→ Q

What are Types of Hidradenitis??

HL

- ① . Apocrine Hidradenitis
- ② . Neutrophilic Eccrine Hidradenitis
- ③ . Recurrent Palmoplantar Hidradenitis:

يتبين للأطباء وللباحثين في بطن القدم غالباً شكوتهم بتقيص
وجع وعدم القدرة على المشي

lesion : EN like Painful S.C
Nodules at sole

Etiology → unknown but ±
Pseudomonas

Recurrence & predisposit-

- Wet shoes
- Cold
- damp weather

: → NSAIDS

Rheumatoid Neutrophilic Dermatosi

HL

- Papules, Nodules & plaques sometimes ē Annular
Morphology affecting patient ē Active R.A
& ↑ R. Factor
- usually at face & Extremities.

• ## : spont. resolut- or by ## of R.A.

(320) للشيخ (بولونيا)

SAPHO Synd.

(one of Sero-ve spondylo-
arthropathy)

- Synovitis : ant. chest joints. & axial skeleton.
- Acne
- pustulosis
- Hyperostosis
- Osteitis

24

2. Infections Caused by Staph. Exotoxins.

Organism: Staph. aureus (phage gp II, Type 71)

Toxins: 3 Types

epidermolysis → I. Epidermolytic Toxins

A, B₂D (Exfoliatins)

- Bullous impetigo
- SSSS

II. Enterotoxin B & C.

50% of Non-MTSS
(Menstrual Toxic shock synd)

Non-genital source of staph

(PVL) Pantan-Valentine Leukocidin
in CAPRS
Leukocyte destr. & Necrosis
Toxic Shock Synd Toxin

III TSS T₁

MTSS (50%)
Scarlatini-form Eruptions
Toxin-Mediated Perineal Erythema
recurrent

Staphylococcal scalded skin

Synd (SSSS)

1st degree burn

25
14.18

Reiters

(Riters dis & Lyell's disease & pemphigus Neonatorum)

Age: usually, infants & children.

rarely: Adults (d-t renal EXcretion of these toxins) EXcept if they have CRF or Immuno supp.

Mortality rate ↑↑

Pathogenesis: (Staph. aureus phage gp II Type 71)

Exfoliative Toxins A, B, D

may

Target DSG1

Act as Super antigens

++ T Cell cytotoxicity

Both

Acantholysis

CIP * Staph focus ±

Conjunctivitis

OM

occult Nasopharyngeal inf.

Septic focus

1. General Constitutional manifest. (Prodromal symptoms)

Fever

Irritability

Sore throat

Rhinorrhoea

Severe skin Tenderness = scalded erythema

(FAHM)

any erythema taking large area

called

Erythroderma

2. Tender Erythema

First localized to head & Neck & Flexures

1-2 hrs

Generalized - (Erythroderma)

Bullae (large, flaccid +ve Nikolsky Sign)

↓
3-5 d^s

④ Sloughing = → raw skin & areas of thin varnish like crust leaving behind moist

level of separation is on granular cell layer while basal cell layer is intact → heal w/out scarring.

⑤ Reepithelialization (Healing) is in 1-2 wks without scarring.

Face → facial Edema, Sad man facies, perioral crust & Red. furrow

- Complications
- Alopecia (TE)
- Onychomycosis
- (RF)
- Vocal Cord Paralysis
- Carpal Tunnel Synd.
- Amenorrhoea

Mortality Rate = MR

- 3% in children
- 50% Adults (Healthy)
- 100% Adults w/ underlying dis.

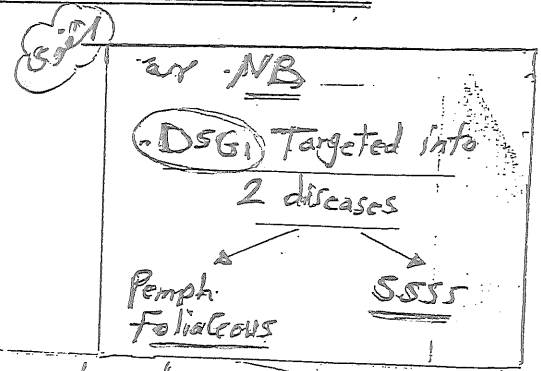
• Clinical Varieties:

① Localized SSSS (Bullous impetigo)

② Generalized SSSS

③ Abortive SSSS (Scarlatiniform): only show Erythroderma & desquamation w/out Bullae

+ve Dermal infect.
↓
-ve Dermal infect.



• Diagnosis

① Clinical

② Staph Isolate

③ Toxins detect

From { Eye, Nasophary, Feces, Skin }
Pyogenic inf.

Blood: -ve in children (but) ±ve in adults

✓ Frozen sect. then ELISA or double Immuno diffusion.

DD

SJS

1- TEN: احمرار

- 2- Sun burn,
- 3- drug Erupt~
- 4- Kawasaki
5. Extensive bullous Impetigo.

حالات

امراض التي فيها

- ①. Nikolsky sign
- ②. Franck smear

SJS/TEN

SSSS

AET

Age

Skin

MM

Face

Nikolsky sign

Healing

Path

Zanck Smear

TH

Prognosis

- usually drug induced
- Adults > 46 Ys.
- Areas of sparing present
- involved (Severe)
- Lip & mm redness & oedema
- in some areas itis, X
- difficult to Elicit. (±)

> 14 ds (scarring)

Derm-epid. Separatⁿ (DET)
Sparse dermal infilt.

- * Cuboidal cells @ Large Nuclei (of Lower Epid.) or
- * Inflammatory cells.

- 1- Burn Unit
- 2- (IVIG) ✓
- 3- CS (Controversy)

Bad (MR ??)

- Staph aureus exotoxins
- infants & young children
- Generalized ✓
- Uninvolved X
- Perioral crusting & fissuring
- mild facial swelling & Erosion.
- +ve in seemingly uninvolved skin. (++)

With in 7-14 ds e or e out th

Split at granular layer
No dermal inflamm. infilt.

- * Elongated Epith cells @ Small Nuclei (upper dermis)
- * No inflammatory cells.

- 1- Antibiotics ✓
- 2- Supportive care

Good. (MR) ??

① Antibiotics (Beta lactamase resistant) for 1W

② Emollients.

③ TH of staph Carriers.

"NB"

Hospitalizatⁿ & IV TH → For Extensive, Generalized cases
oral, Home TH → For mild cases.

Toxic shock Synd (TSS)

Def: Multi system dis. Caused BY St aureus producing Exotoxins.

Types:

	Menstrual TSS (MTSS)	Non MTSS
<u>Source of Staph</u>	Vagina of Menstruating Women using high absorbant Tampons مانقان الامني	<ul style="list-style-type: none"> Surgical procedures. Wounds, Ulcers Catheters. IUD
<u>incid</u>	Was Common at 1980, Now less Common	more Common Nowadays.
M:F	Only F ف و بس	M=F
<u>Causative Toxins</u>	TSS T ₁	<ul style="list-style-type: none"> TSS T₁ Enterotoxins B & C.
MR	5%	12%

Etiopathogenesis: the Major risk factors is absence of Antibodies against TSS T₁ → 3 Mechanisms:

(SSSS) ^{usil}

- ① direct toxic effects on Multi-organs.
- ② Impaired Clearance of Endogenous Endotoxins derived from Gut flora.
- ③ Act as Super Ag.

CIP & Criteria for D:

• Fever $> 38^{\circ}\text{C}$

• Rash: diffuse macular erythema

• Desquamation: after 1-2 wks (sp. palmo plantar)

• Hypotension: Systolic $< 90\text{ mmHg}$ for Adults (< 5 Percentile for children)

• Involvement of ≥ 3 organs:

- CNS (disorientation)
- Renal (\uparrow Pus, BUN & Creatin.)
- GIT (Vomiting & diarrhoea).
- Liver (\uparrow enZs).
- Blood (Thrombocytopenia).
- MM (Hyperemia)

• Lack of evidence of other causes:

(-ve)

◦ -ve Serology for: RMSF & leptospirosis

◦ -ve Culture for: Blood, CSF & throat. isolation

toxins

NB

• Streptococcal TSS differs from Staph TSS in the following:-

1. rapid progressive
2. more destructive
3. MR 30%.
4. Strept isolation (GA, M strain)

Treatment

Shock H.
&
Antibio.

1. Antishock Measures e.g fluid for Hypotension
2. Source of inf. (remove meshes)
3. Antibiotics (Beta lactamase resistant): antibiotics that eradicate the toxins are preferred e.g rifampicin, clindamycin, Quinolones.
4. if severe shock unresponsive antibiotics \rightarrow low dose Cs

① Diseases Caused by Streptococcus

(28)

(Tend to Invasde the skin
proper)

1- Direct infection:

✓ [Impetigo (non Bullous)
Ecthyma

✓ [Cellulitis
Erysipelas

→ Intertrigo

NB!

W/L 2 [Perineal Dermatitis.
Angular Cheilitis.
Blistering distal dactylitis
Necrotizing Fasciitis

. strept. ulcer

2. Secondary infection of ulcers Burns Eczema

3. Toxins :-

[Scarlet fever. (Erythrogenic Toxin A, B, C)

[Toxic shock-like synd. (Exotoxin A, B) ← more severe than
TSS of staph

4. Reaction to bacterial Ag:

↳ EN ← Erythema Nodosum.

↳ Vasculitis (Henoch-Schönlein purpura)

↳ Rheumat. Fever (EMF)

5. Diseases Influenced or provoked by it:

. Psoriasis (Guttate).

impetigo & Ecthyma → See staph inf.

Cellulitis & Erysipelas: inf

Cellulitis

Def → infection of deep dermis & S.C.T Caused most commonly by GABHS & S. aureus. (Strept > Staph)

Causative organism: → Streptococcus + Staphylococcus

• Most Common → Strept ($\frac{2}{3}$) & Staph ($\frac{1}{3}$)

• Less Common: G-ve bacteria e.g.

• H. influenzae

• G-ve organisms

• Atypical Mycobact.

• opportunistic fungi

in immo compromised

Cryptococcus

Aspergillus

inf. inf

T. pedis is 1 common

Route of Infection:

• Immuno Competent → Exogenous (skin break) route

• Immuno Compromised → Haematogenous (Blood)

Xerosis

Predisposing factors:

- Lymphoedema
- Alcoholism
- DM
- IV drug abuse
- PVD

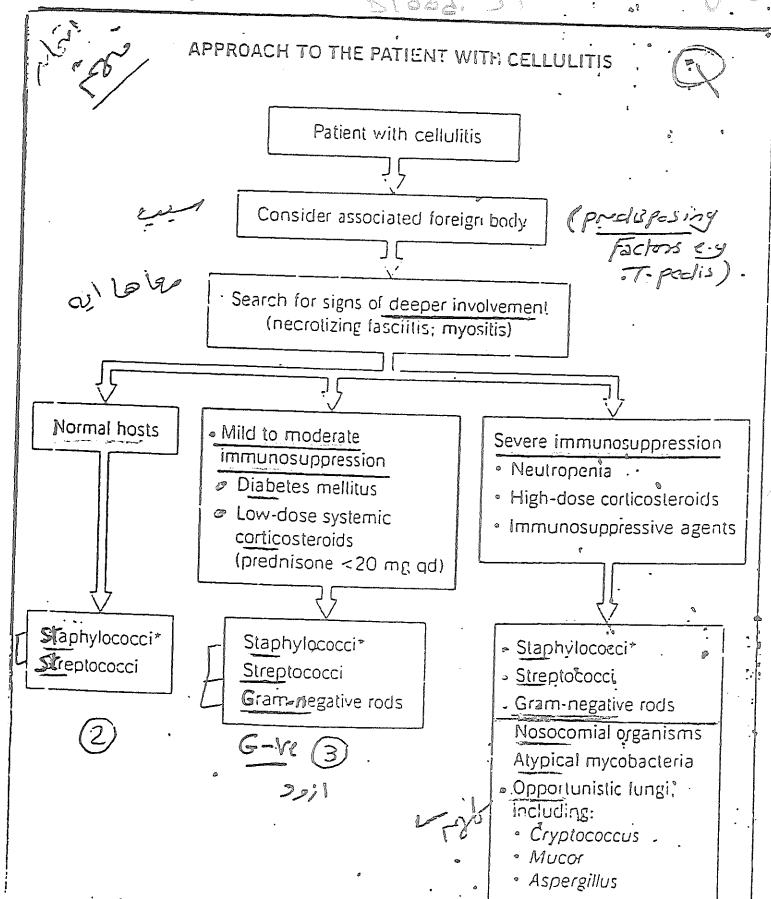
Peripheral Vascular Dis.

• Lymphatic damage

• T. pedis

Causes of Recurrence:

1. T. pedis
2. obesity
3. Lymphatic damage
4. Venous insufficiency



CIP ① Systemically: FAHM

② Locally: ① 4 Cardinal Signs of inflamm.

- Hotness
- Redness
- Tenderness
- Swelling

ss. ed. age
↓

② the lesion has ill defined & Non palpable border

there ±

③ severe inf. → Vesicles, bullae, pustules & Necrosis (Bullous Cellulitis)

④ Ascending Lymphangitis & regional L-N may be +ve

⑤ Site: Commonest Site in

Adults
↓
lower extremities

Children
Face & Neck

Iv drug Abusers
Upper Extremities.

Facial Cellulitis
G-ve H. influenza
G-ve Ab. de. 2

Complications:
(rare)

① GN (if Nephritogenic strept.)

② Endocarditis.

③ Lymphadenitis.

④ Lymphatic System damage →

Recurrent attacks

Pathology: mild to mod inflamm. infect. (mainly N & L)
Through dermis & extend to S.C.T

oedema & dilatation of Lymphatics & BVs

Spec. stain → for organism detection

subepid. bulbe. (±).

DD: 1- Erysipelas

2. Erysipeloid (Infectious & inflammatory)

3- Pseudo Cellulitis: Inflammatory (Non infectious) Cellulitis Like lesions.

برلنيا لجز ٢
(p 1084)

نقش

NB: Erysipeloid = pseudo cellulitis

Invs

- ① WBCs: usually NL or slightly ↑↑
- ② Blood culture → usually +ve in Immunocompromised.
- ③ Need for invs. is:

H. influenzae

children & Immunocompromised

- ↑ WBC (← left shift)
- ↳ +ve culture (Blood)

- ① Needle Aspiration
- ② Biopsy

Treatment: ① Mild cases: at Home tt by antibiotics (anti stapt & staph) for 10ds.
MSSA MRSA

- Bed rest
- leg Elevat.
- Wet dressing to bullae & exudate
- T. pedis tt

Antibio-tic

② Severe cases & Facial Cellulitis: Hospitalization &

"Parenteral Antibiotics"

Best Δ antibiotics
 - Unasyn (Cover GABHS & MSSA)
 - Mino cycline or clinda. (Cover MRSA)
 - Flagyl (Cover G-ve)

③ Failed response for 1-3 ds: → Culture & Sensitivity
→ Consider MRSA

④ tt of Recurrent attacks (see below)

⑤ Surgical intervention of: Crepitus, Circumferential or Necrotic.

NB Avoid NSAIDs: may mask symptoms of Necrotizing infection.

NB: Facial Cellulitis Common in

- Elderly > 50 → Staph. or Strep.
- Children → H. influenzae B & S. aureus (Commonest)

Cellulitis d.t:

• Cat & dog bite → Pasteurella Multocida

• injury during water contact → G-ve

• Infant Cellulitis: Consider Hematogenous route
(< 6ms) neonatal → Group B Strep. → Septic focus: osteomyelitis, Arthritis, Kidney, ...

• Immunocomp. → G-ve or Fungal

• peri wound < 24 hrs → Clostridium causing

Cellulitis	
acc. to Age	
Infant < 6ms	
up to 6ms	Staph & Strep.
Children:	
6ms - 12ms	Staph & Strep.
12ms - 18ms	H. influenzae
18ms - 24ms	Staph & Strep.
24ms - 30ms	Staph & Strep.
30ms - 36ms	Staph & Strep.
36ms - 42ms	Staph & Strep.
42ms - 48ms	Staph & Strep.
48ms - 54ms	Staph & Strep.
54ms - 60ms	Staph & Strep.
60ms - 66ms	Staph & Strep.
66ms - 72ms	Staph & Strep.
72ms - 78ms	Staph & Strep.
78ms - 84ms	Staph & Strep.
84ms - 90ms	Staph & Strep.
90ms - 96ms	Staph & Strep.
96ms - 102ms	Staph & Strep.
102ms - 108ms	Staph & Strep.
108ms - 114ms	Staph & Strep.
114ms - 120ms	Staph & Strep.

Erysipelas

(St Anthony's Fire)

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Def: streptococcal inf. of Whole dermis ^{"± upper S.C.T"} ± significant lymphatic spread.

NB: Very similar to Cellulitis but differs in:

- affect ! Whole dermis. (No S.C.T affect)
- Caused by: Strept. (Other organisms are rare)
- lesion char by:

- Well defined raised border
- ± dimpled (peau d'orange)
- ass. ± Lymphangitis (streaks) ^{also lesion in NP}

Note:

Chr. Recurrent Cellulitis & Erysip.

Once Cellulitis or Erysipelas

Single attack of Cellulitis & Erysip.

usually followed by

Recurrent attacks ^(Always.)

① 1- # of predisposing Factors e.g. T. pedis, Lymphoedema.

② Chr. Antibiotic Use e.g.

- Long acting Penicillin ^{"D-6-azap"}
- Penicillin G (250 mg bid.)
- Erythromycin (250 mg once or Twice / day) ^{For life}

if allergic give

DD of Erysipelas Like (Erysipoid)

inflammatory like
"Non infectious"

or Cellulitis like (PseudoCellulitis)

Conditions:

Erysipoid of Rosenbach

Cancer Erysep-
Episepoid

Angioedema

EN

bites (insect & spider)

CD (Contact Dermatitis)

Drugs $\left\{ \begin{array}{l} FDE \leftarrow \text{Fixed Drug Eruption} \\ TEN \\ Warfarin Necrosis \end{array} \right.$

دوپلر سونوگرافی
Doppler

DVT & lipodermatosclerosis

Sweet's & Wells Synd.

HZ

ECM

FMF

Inflammatory $\left\{ \begin{array}{l} \text{Morphea} \\ \text{GA} \end{array} \right.$

NB

Group B Streptococcal inf.:

25% of Adults having it as a commensal
in Genital & GIT.

inf. Caused by it \pm :

- Post partum abd. & Perineal Erysipelas
- Neonatal Sepsis & meningitis
- orbital & Facial Cellulitis

Streptococcal Intertrigo

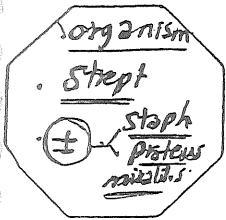
under recognized entity

usually affect moist intertrigenous areas (axillae, Neck, groin, antecubital areas & Post auricular) of infants & children (d.t moisture & Friction) ← منبؤنا كثر

CLP: ① Well defined finely red Erythema & ± Psoriasiform plaques. (but No Satellites)

② Ass ē $\left\{ \begin{array}{l} \text{Pain} \\ \text{Bad odour (chic)}^* \end{array} \right.$

?? Fungal



DD: other Causes of Intertrigo:

- 1- ps ←
- 2- SD ← Seborrheic dermatitis
- 3- Simple (Treated by ↓ Friction & moistures; but if No response → Consider Strept inf.)
- 4- Other causes (See Fungal inf.)

- ③ ① oral penicillin for 10 ds $\xrightarrow{\text{if No result}}$ Consider G-ve mixed inf. (proteus)
② Topical Antibiotics + weak Cs.

NB

perineal Dermatitis = Perianal Cellulitis

(Streptococcal Perianal dis.)

- GABHS

- Age: usually bet. 1-8 Ys (usually < 4 Ys)

- Clinically: it is Dermatitis rather than Cellulitis.

• infs usually: preceded by pharyngitis (so always be considered in patients ē Guttate PS).

• perianal Erythema (2-3 cm) around the anal verge ass. with tenderness → painful defecation → retention, Bloody streaks, Soiling of clothes.

NB

penis & vulva ± affected.

DD → See Perianal pruritus. (or) Napkin Rash.

- ③ → ① Antibiotics (2-3 wks) then perianal swab to detect Nephritic seric str.
② Topical Antibiotics

Blistering distal Dactylitis

35

Age: Typically 2-16 Ys

± Non-fled digits.

CIP

Tense superficial blisters on a tender Erythematous base over the Volar fat pad of phalanx of finger or thumb & ± Toe.

organism: Strept or Staph ^{Aureus} _{Epidermidis}.

- ##: ① incision & drainage ② Topical antibiotic
③ Systemic antibiotics (prevent New inf or extension of inf.).

Toxin mediated
Strept inf

Scarlet Fever (Scarlatin)

AET: GABHS Erythrogenic toxins A, B & C → DHR

Age: 1-10 Ys (after 10 Ys; 80% developed Antibodies.)

- HX: ① usually: following Tonsillitis or pharyngitis
② may follow surgical wounds (surgical scarlet fever)

CIP

Age: 1-10 Ys

HX: Tonsillitis or pharyngitis

Prodromal
symptoms →

FAHM, sore throat, abd. pain & Vomiting

Rash: start after 1-2 ds; at the

Sandy rash

Neck
chest
axillae

4-6 hrs

Involve the whole
Body

Ch B Y: Tiny papules on Erythematous backgrounds
(Sunburn & goose pimples)

خارجی

Flushed cheeks (slapped cheeks)

Circumoral pallor

Tongue: at first white & bright red papillae →
then Beefy (straw berry).

Postia's Sign: linear petechial ^{purpuric rash} streaks at axillary,
inguinal & antecubital creases.

Desquamation: after 1-2 wks & continue for 2-6 wks at Hands & Feet

(Isotretinoin) → dryness or chapped lips (Staph, Strep, Candi)
Angular Cheilitis → See Candidiasis (3)

Necrotizing Fascitis

(HIV قىرىقۇش) ←

Def: Acute (streptococcal) Necrotizing inf. involving the Fascia

- Source of inf. 1. Denovo
- 2. Following Surgery or Perforating Trauma or Cellulitis.

organism: many be involved:

- Strep. ✓
- Staph
- enterococci
- Pseudomonas
- Bacteroides.

→ So 2 Types

Type I → polymicrobial (90%)

Type II → streptococcal (10%)
= unimicrobial

staph & strept.

Cellulitis ↓ CIP

1-2 ds after Entry → Tense spreading

redness, Pain, oedema → Central patches of blue-dusky discoloration

with or without Serosanguineous blisters (bullae)

Anaesthesia of involved skin is Very Chic

Severe Systemic Toxicity: Fever, chills, Malaise, shock & ↑ WBCs.

4th-5th d.:

purpule areas become Gangrenous

ulcers

III → 1. Early surgical intervention

NB Signs that aid in delineating the extent of deep involvement include:

- 1. Hypotension → severe infection (toxic shock)
- 2. WBC > 15.4
- 3. Na serum < 135 mmol/L (Hyponatremia)
- 4. MRI.

2. IV antibiotics

3. IVIG: ± useful in Type III.

8/5/19

Streptococcal TSS

(Strept. toxic shock like synd.)

Def.

Rapidly progressive, often fatal illness

Caused by GABHS commonly presented with:

- Fever
- Shock
- Multiorgan Failure
- Soft tissue inf.

→ (M Type 1 & 3)

Etiopathogenesis:

Source of Entry $\left\{ \begin{array}{l} \text{Unknown ?? (50\%)} \\ \text{disruption of cut. barrier} \end{array} \right. \rightarrow \text{Streptococcal entry (M Types 1 & 3)}$

→ release of Exotoxins A & B → shock &

Tissue injury by 2 mechanisms:

① As Super antigens (bound to MHCII on APCs & V β region on T cell R $_2$)

→ TNF α & IL1

② Formation of M protein / Fibrinogen Complex.

NB: IL6 play a crucial protective role via \downarrow TNF α

Epidemiology

Age: 20-50 y.

Sex: M=F.

CIP

① Pain: Severe local pain in extremities is the most common initial symptom with or without (50%) signs of soft tissue inflammation (4 signs)

② Violaceous hue, bullae or necrosis → indicate deeper inf. (Necrotizing fasciitis or myositis) → Bad Prognosis

ppd 0.1 ml \leftarrow ③ Non specific flow like symptoms: Fever, chills, myalgia, diarrhoea, ~~fever~~

- ④ Shock & MOF after 2-3 d
- ⑤ Severe Complications may occur:
 - [RDS
 - [RF
 - [DIC
 - [Death (30-60%) [MR].

Case Definition of Staph TSS
(Criteria For S)

- Isolation of GABHS from NLLy Sterile site (Blood, CSF & Biopsy).
- or
- Isolation from non sterile site (throat, sputum, vagina)
- and
- Hypotension: SBP < 90
- and
- ≥ 2 of the following signs:

- Renal impairment
- Coagulopathy (PLT < 100,000) D/D
- Liver Impairment
- RDS
- Generalized macular Erythema
- Skin < (±) desquamation.
- Soft tissue Necrosis.

- DD
- TSS (Staph. TSS differs from Staph TSS in)
 - USSS
 - Kawasaki
 - Early TEN
 - RMS
 - older Age 20-50
 - rapid progressive
 - severe
 - +ve B Culture (S)
 - MR 30-60%
 - extremity pain
 - Soft Tissue Necrosis

	Staph. TSS	Staph. TSS
<ul style="list-style-type: none"> Age Etiology (Source) 	<ul style="list-style-type: none"> 15-35 Menstrual & non Menstr 	<ul style="list-style-type: none"> 20-50 y. Unknown or Skin break, varicella, Bruises & Bullae.
<ul style="list-style-type: none"> Pain in Extremities Diffuse Macular Eryth. Vesicles & bullae Soft tissue inf. 	<ul style="list-style-type: none"> - Very Common rare rare 	<ul style="list-style-type: none"> Common less common less common (5%) Common
<ul style="list-style-type: none"> Hypotension RF. 	<ul style="list-style-type: none"> 100% Common 	<ul style="list-style-type: none"> 100% Common
<ul style="list-style-type: none"> Blood Culture +ve MR. 	<ul style="list-style-type: none"> < 15% < 3% 	<ul style="list-style-type: none"> > 50% 30-60%

Skin
manifests
are marked
in Staph
TSS.

- (H) [1] Supportive Ht For Shock → IV fluids & Vasopressors.
- [2] Clindamycin: → -- bact toxins, (1st choice)
- [3] Early surgical intervention eg drainage, debridement, Fasciotomy, amputation.

Skin diseases caused by Coryneform bacteria

40

عن

- def. { G+ve
non spore forming
Rod shaped.

Erythrasma

Def chr. (superficial) skin inf.

Caused by *C. Minutissimum*.

(G+ve, non spore forming, Rod shaped)

Predisposing factors:

- obesity
- DM
- Excessive sweating & maceration.

خارجي الوس
جافين في
Axillae

CIP →

3 Varieties

1. Classical form: (Colonial)

- 2nd most common.
- at flexures: groin, Intergluteal & inframammary,
- patches chr by
 - Well defined
 - Finely scaly
 - pink - brown (hyperpigmented)
 - slowly spreading

axillae,
intercubital

2. Toe web inf. →

- Most common.
- bet 5th & 4th or 3rd & 4th Toes
- Scaling, fissuring & maceration.

تكرري در فترتي

may be confused w organisms in these
areas: (Dermatophytes, Candida, G-ve, staph. au.)

Skin diseases related to coryneform bacteria

- AV → propionibacterium acnes & P. granulosum.
- Erythrasma → *C. minutissimum*.
- Trichomycosis axillaris → *C. Tenius*
- Pitted keratolysis

5 others:

- Cut. ulcer w *Cory. diptheriae*
- JK group. *Corynbact* → Commensal
- Arcanobacterium infantum*
- Hemolyticum*.

6 shunt tube infected

[4] Generalized form: (Trunk & extremities): (disiform) (41)

- least common
- Common among middle Aged black women
- Patches (as in classic form) at trunk & proximal Ext.

(Diagnosis) → W.L Examination: Coral Red Fluorescence
(pink-orange)
" d.t Coproporphyrin III "

(Treatment)

[1] Systemic: Erythromycin 250 mg / 4 times / d for (1w)

[2] Topical: (a) Antifungals — AZoles
Tolnaftate
Miconazole

(b) Antibiotics — Fusidin
Erythromycin
Clindamycin.

تفتيح البشرة
" ج. م. م. م "

(NB) • Causes of pink fluorescence by W.L:

1. Follicular openings of NL skin of face & Trunk (?? Propionibact).
2. Some Necrotic Tms
3. AV
4. Tongue
5. AN: in groin & axillae.

DD: See DD of T. Cruris.

Trichomycosis Axillaris (Trichomycosis Nodosa)

(42)

Def

Bact. inf. of Axillary (less commonly pubic hair)
Hair by Corynebacterium Tenuis (G+ve, Rod, non spore)
Ch-BY: presence of yellow, red or black concretions on Hair shaft.

في ايلجيس

CIP
(3)

① Concretions: Surrounding the hair shafts → Beaded appearance & yellow, red, black.

② Sweating is Colored → stain clothes (Commonest complaint)
↓
Malodorous. (Same color of concretions)

NB: DD: "Chromhidrosis" to diff. (10% KOH + injected Hair)

② Beaded Hair (See Hair).

① Clipping of Affected Hair

② Antimicrobials: Erythromycin, Clindamycin & panoxyl.

③ Anti perspirants: "عرقا"

show the bact. in the concretions (Coryneb.)

في ايلجيس
Wart

(EM 2010)

→

Pitted Keratolysis

Crateriform
Variant: no pits but the whole foot under MTP joint

Def: Superficial skin inf. Caused by Coryn.

Causative organisms: many ← Micrococcus (Kytococcus) tendentarius
Dermatophilus congolensis
Corynebact. / Actinomyces / Streptococcus

① Minute superficial pits & Erosions in St. Corneum (1-5 mm); usually at pressure areas of Sole (Heel & Toes). [d.t proteases produced by Bact.]

② Malodour [d.t sulfur product].

③ Hyperhidrosis (Common but not essential).

Palm ± affected
↓
Callus rather than pits.

HT: (Topicals) ① Antibiotics

• Fucidin
• Erythromycin
• Clindamycin
• Benzoyl Peroxide

② Antifungal

• Miconazole
• Clotrimazole
• white field

③ Anti-perspirant

↓
Cura

Self limiting

Erysipeloid of Rosenbach

• def. Acute infection of skin & other organs caused by the microorganism Erysipelothrix rhusiopathiae (E. insidiosa)

• organism: Gram +ve, non motile, rod shaped, filament producing. Commensal pathogen in Mammals, birds, Fish

• Risky pt. those dealing with fish or meat of infected animal, poultry or shellfish.

• So Farmers, butchers, Fishermen, Vet. Surgeons all are at risk.

• CIP (3) Varities Localized cut. [Erysipeloid of Rosenbach]
→ Generalized
→ Systemic

(A) Localized Cut. [Erysipeloid of Rosenbach]

• IP (3 ds) after inoculation by prick penetrates by fish, mammals or bird bones → Trauma

• Localized to area around the inoculation

• Localized, Erythematous bluish patches

That expand slowly & have well defined border 2-4 w. → Spontaneous resolute → Fast deq.

• Commonest areas: hands, finger webs, Forearms
• ± ass e.: Pain, Tenderness, oedema, blistering

(B) Generalized (diffuse) cut.

• Generalized pink - violaceous lesions with advanced edge
• ± systemic manifs

• -ve Blood Culture

(C) Systemic

• ass e. systemic manifs & Multiple organ affect
• +ve B. Culture

• Self limiting in 2-4 w.
• penicillin 1gld for 5-10 ds
• Erythromycin, Klaramycin, Tetracycline

• B. Why antib. less its self limiting?

• prevent dissemination accelerate the healing

Actinomycosis

Def: Subacute to Chronic Bact. Infection ca by:

- Clinically:
- Suppurating Abscesses
 - Granulomatous Inflamm.
 - Sinus formation
 - Sulphur granules

Can affect skin,
bone & int. organs
↓
" Mycetoma like
lesions

Caused by: Actinomyces Israelii. (also A. bovis)

- Gram +ve
- Anaerobic ~~or~~ microaerophilic
- Non acid fast.
- branching or Filamentous

Also
(in soil)

→ NL inhabitant of — Mouth, GIT, genital Tract.
(So inf. occurs by contagious
Endogenous spread).

Clinical Features (Types)

- ① Cervicofacial (50-70%)
- ② Thoracic & Pulmonary (15-20%)
- ③ Abdominal (10-20%)
- ④ primary cut. (uncommon)
- ⑤ pelvic
- ⑥ Lacrimal

Epidemiology

- Age: any age but commonest 20-50 y.
- Sex: ♂ > ♀.

① Cervicofacial Type (Lumpy Jaw)

(49)

• Most Common type (50-70%)

• Hx & Source of inf:

• Bad oral hygiene

• Dental Caries.

• Orofacial injury or procedure

Periodontal Abscess
→ periosteitis &
osteomyelitis →
Spread to skin.

• lesion ch by

• Site

Mandible > Maxilla

• Start as bluish area at mandible (Jaw angle)

→ Nodules that's Erythematous,
at first Tender Later painless & woody

→ Formation of Fistulous abscesses that
drain purulent material with cheek
(yellow) sulfur granules (clumps of bacteria)

• No ass L.N

• ± FAHM.

• ± Extension to < Maxilla
orbit

② Pulmonary Type (Thoracic):

• 2nd most Common (15-20%)

• Hx & Source:

• Recurrent aspiration of infected oral
material (Epilepsy).

• Lesion ch by:

• pulmonary Cavities at lung bases →

Chest Symptoms (Cough, dyspnea, ...)

• Extension: to pleural → Chest skin →
multiple draining sinuses & Pleurocut. Fistulae.

③ Gastrointestinal Type:

• less Common (10-20%)

• Hx & Source

• may occur spontaneous or following inflammatory
Bowel disease or surgical procedure or ingest

CIP: . Non Specific Constitutional manif.

Commonest areas
↓
appendix Caecum

. Manifs of appendicitis.

. Granulomatous affection of Bowel → appendicular mass → spread to the skin.

Extension: . liver → jaundice

. Abd. wall → skin lesion

. ovaries

. Kidneys.

④ pelvic Type:

. History of IUCD.

. lower abd discomfort, menorrhagia

. pelvic mass.

⇒ No Cut. lesions

Diagnosis

Sulfur granules

- . 1-2 mm
- . yellow
- . adherent to gauze dressing.

Mic: narrow bacillary forms & elongated Hyphae occasional branching.

Culture (difficult)

"Heart-brain Blood inf. agar"

. Anaerobic

. Enriched at:

37°C for 2-4 d

white glistening irreg. Colonies.

Histopath. (below.)

Pathology:

. at first → suppurative (intense Neutrophilic infiltrate)

. Later → granulomatous

Center: Abscess with "granules"

Periphery: . Histocytes
 . Epithelioid Cells
 . Giant cells

Granules ch. by:

(46)

at low magnification

↓
Cauliflower like

(Branching
filaments)



at higher magnification

↓
delicate branching

filaments at the periphery
of which show Eosinophilic
clubs composed of
Immunoglobulins (Splendor-
Hoepli phenomenon). They
resemble rays, hence the
Name (Ray fungus).



Other invs,

• CBC: anemia & leukocytosis

• ESR: ↑

• CRP: ↑

• CXR

• CT

##

Rx

① Penicillin G 10-12 mill. Uld
(IV)

→ Surgical
Excision

→ Penicillin 2-5 Mld
(IM)

② Other antibiotics: Tetracycline

• Erythromycin

• Chloramphenicol

• Recently: Imipenem / Cilastatin

(Cover actinomyces & Companion
Bacteria)

Erudicina

Helicobacter pylori Inf. & Skin diseases

Def. G-ve, microaerophilic bacterium that can inhabit various areas of stomach specially the antrum.

Disease caused by it:

A. GIT diseases: Gastritis, PU & Carcinoma

B. Cut. diseases: may have role in:

- AV
 - Angiodema & chr. urticaria.
 - Atopy
 - Behcet
- LCV
 - Nodular prurigo
- Perioral Dermatitis
- Rosacea
- SS
- SSc
- Sweet Synd.

Diagnosis: 1. H. pylori fecal Ag test. وسيفي -ve بعد العلاج، لذا استخدم للتأكد من العلاج

2. Carbon 13 Urea breath test (UBT)

المريض يشرب يوريا + Carbon Isotope + بعد فترة
ينفخ مركب ال Carbon Isotope في أنفاسه (يأخذ
(Urea splitted by H. pylori urease enz.)

3. H. pylori abs (IgG) لا تستخدم لتأكيد فعالية العلاج

4. Antibiogram: in areas where is resistance to Clarith. & Metronidazole

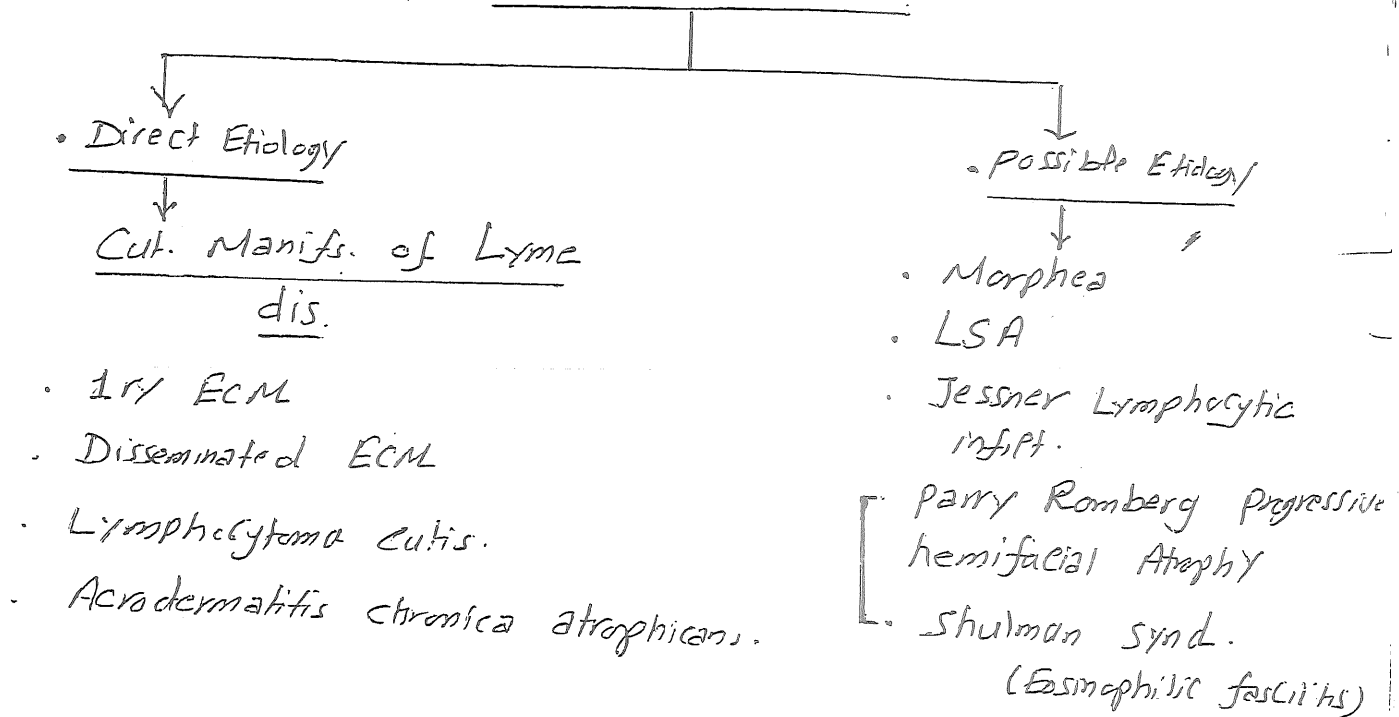
Treatment → Triple therapy for 10-14 d.

(OAC) [Proton pump -- (omeprazole)
Amoxicillin (if sensitive) → Metronidazole
Clarithromycin]

(BMT) [Bismuth sub-salicylate
Metronidazole
Tetracycline]

Def- & Introduct → see Lyme dis.

Role in skin diseases



NB Types of Syphilis:

1. Syphilis barbae/Vulgaris.
2. Lupoid (Staph, granulomatous destructive)
3. Mycotic (Zoonophilic Fungi).
4. Herpetic.

31-7-14



• Role of Super Antigens in SKIN

diseases (Toxin mediated skin diseases)

Def. of Super Ag: Non antigen Toxins (proteins) secreted by Staph. & Strept. → Stimulate T-cell → Secrete massive amounts of cytokines → severe systemic Manifs.

• Difference bet. Antigen & Superantigen:

Superantigen differs from Antigen in:

1. No need for processing.
2. Not placed on MHC-II (of APC) Groove but placed directly at any place, but
3. Interact directly w/ variable region "V β " of TCR, (on T cells)
4. More potent stimulant of T-cells (excite 5-30% of T-cells compared to $\approx 0.01\%$ stim. by Ag) → massive cytokines release: IL1, IL6, & TNF →

- Fever
- Hypotension
- Shock
- organ damage
- Skin rash (Scarlatiniform or Morbilliform)
- PP Erythema & Edema
- Strawberry Tongue
- Conjunctival inject

(Severity of Condition depends on $\left(\begin{array}{l} \text{Toxin dose} \\ \text{Route} \\ \text{Immunological status} \end{array} \right)$)

- NB ① Sulphadiazine Creams: if not in therapeutic dose → ↑ toxin product
- ② NSAIDs: ↓ Immunity & ↑ toxin product

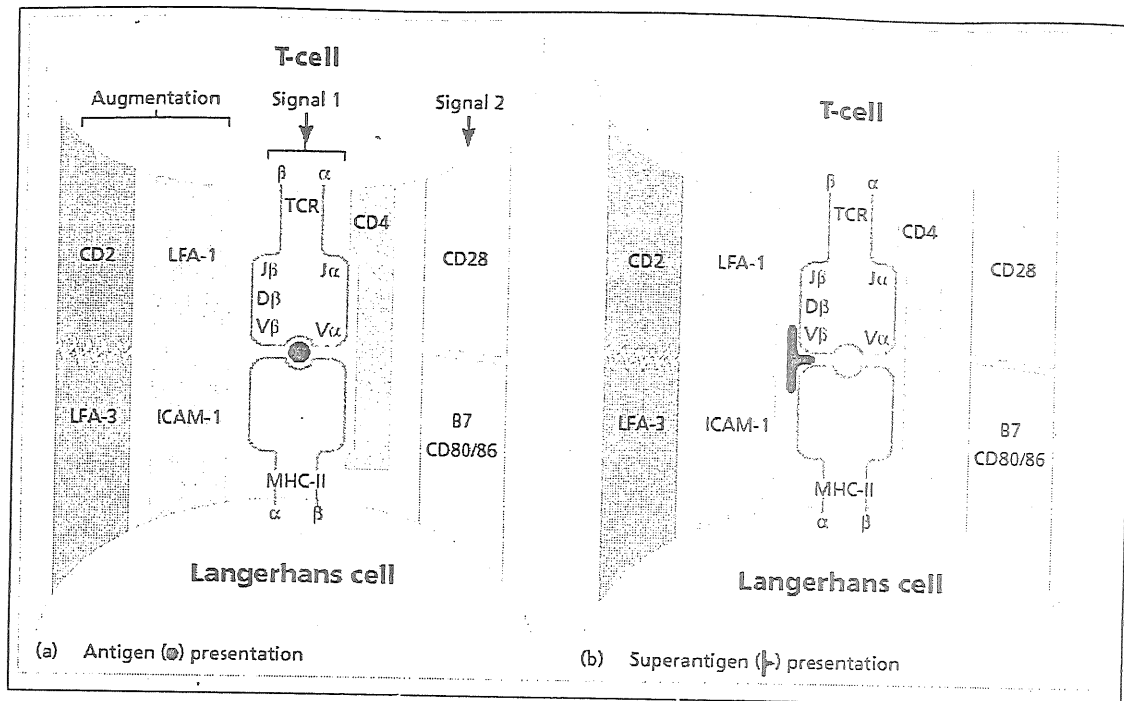


Fig. 2.12 T-lymphocyte activation by (a) antigen and (b) superantigen. When antigen has been processed it is presented on the surface of the Langerhans cell in association with major histocompatibility complex (MHC) Class II. The complex formation that takes place between the antigen, MHC Class II and T-cell receptor (TCR) provides signal 1, which is enhanced by the coupling of CD4 with the MHC molecule. A second signal for T-cell activation is provided by the interaction between the costimulatory molecules CD28 (T cell) and B7 (Langerhans cell). CD2/LFA-3 and LFA-1/ICAM-1 adhesion augment the response to signals 1 and 2. Superantigen interacts with the TCR V β and MHC Class II without processing, binding outside the normal antigen binding site. Activated T cells secrete many cytokines, including IL-1, IL-8 and interferon- γ , which promote inflammation (Fig. 2.13).

• Toxins (Super antigens) Mediated Skin diseases:

- SSSS
- TSS (staph & strept types)
- Recurrent Toxin Mediated perineal Erythema
- Red Synd. (recalcitrant Erythematous & desquamative Synd).
- Scarlet Fever
- Necrotizing Fasciitis
- Kawasaki dis
- Psoriasis
- Atopy (staph)
- Food poisoning

HL — [Strep]

Pseudomonas Skin Inf.

(Andrews)
Emol
DMSO

(51)

- 1. Gram -ve Folliculitis
- 2. Hot tub folliculitis
- 3. Toe-web infection
- 4. Ecthyma Gangrenosum
- 5. Green Nail Synd.
- 6. External Otitis
- 7. Blastomycosis like pyoderma

1. Gram -ve Folliculitis → See Acne vulgaris.

Spa
pool
folliculitis

2. Hot-tub Folliculitis (Pseudomonas Folliculitis):

EMed 2009

predisposing
Factors

1. Minor Trauma

أضرار جراحية
الجلد

2. Hot Water

3. pH > 7.8

4. low chlorine
< 0.5 g/ml

CIP
Folliculitis
or
Dermatitis

• Pruritic follicular, Maculopapular, Vesicular
or pustular lesions usually at Sides of
Trunk, axillae, buttocks & proximal Extremities
[Spare Face, Neck, Palms & Soles]

• usually (1-4) days after bathing in

Hot $\left\{ \begin{array}{l} \text{Tub} \\ \text{Jacuzzi} \\ \text{Public Swimming pool} \end{array} \right.$

the High temp. →
↓ Free Chlorine
(despite NL total
Chlorine level) →
Colonizatⁿ by
Pseud.

(entry) → Route of inf. < H. Follicles
skin break.

• Colonizatⁿ of bathing suit
may occur so may transmit inf. « bathing
Suit Folliculitis ».

• ± ass. with systemic manif.

• HL « Self limiting in 1-2 wks »

① prophylactic:

- water filteratⁿ, automatic
chlorinatⁿ, Frequent changing.
- Keep Chlorine (Free) at 1 ppm level

Keep water PH at 7.2-7.8

Resistant to
topical
antib.

← ② Active H: (if there is systemic manifest or Prolonged dis.):

- Fluoroquinolones
- 3rd generation Cephalosporines.

NB Pseudomonas Hot Foot Synd: reported in children, ch BY: painful, Erythematous, Planter nodules or pustules after wading in a community pool whose floor was coated in a abrasive grit.

DD: Neutrophilic Eccrine Hidradenitis
(Elev. myeloperoxidase)

3. Toe Web Gram -ve inf.:

interdigital T. pedis (usually) start as dermatophyte infect- only (dermatophytosis Simplex); then with Pseudomonas or G-ve inf. → dermatophytosis Complex.

- ↑ inflammation
- Macerat
- denudat- is purulent serous discharge
- marked, Edema & Erythema of surrounding tissues
- also ±: Painful Calf nodules that recur 11-2 wks.

H ① Dermatophytosis Simplex (Scaling & Peeling): → Topical Antifungal.

② Dermatophytosis Complex (wide spread Erythema, Edema & denudat-)

→ Anti Pseudomonas Antib.:

- 3rd generation ceph.
- Fluoroquinolones.

4. Ecthyma Gangrenosum

(52)

Risk Factors (Gravely ill patients):

→ if's ass. with pseud. bacteraemia

- leukemia
- Severe burn
- Malignancy
- Neutropenia or pancytopenia.
- Severe chronic dis.

sign. CIP: « Tense Vesicles or pustules that surrounded by narrow pink-violaceous

halo » → Hyic → rupture →

round ulcers with necrotic black center

→ site: perineal & gluteal > axillar > Extremities > Trunk

Hyic Pustule
Surr. by Erythema

↓ 12 hrs.

Necrotic gangrenous ulcer
Surr. by Erythema

- Healthy infants may develop lesions in perineal area after antibiotic therapy in conjunctⁿ e maceratⁿ of diaper area.

Diagnosis ① clinically: → classic pustule → ulcer.

② Vesicle Aspirate & culture → G-ve bacilli

③ Blood culture (if ass. Septicemia).

④ SKIN Biopsy → 2 Biopsies For Histopath. & culture.

Treatment



① Early institution of IV antibiotics

Aminoglycosides + Antipseudomonal penicillin (Piperacillin).

+

GMCSF (adjuvant H to ++ myeloid precursors in pts w neutropenia).

② If failed medical → Surgical debridement.

7. Blastomycosis like Pycoderma

- large verrucous plaques & elevated borders & Multiple pustules may occur as a chr. Vegetating inf.

• Most patient may have Immuno suppression (← ^{local or} systemic)

• Causative bact. ± :

- Pseudomonas
- S. aureus
- Streptococci
- E. coli

Others

- Green foot synd.
- Hot foot synd.
- *Septicaemia
- Dry inf. of wounds & Ulcers & Burns.
- Periumbilical inf. of infancy → Foul smelling discharge.

4. Ecthyma Gangrenosum

(5/2)

Risk Factors (Gravely ill patients):

→ it's ass. with pseud. bacteremia

- leukemia
- Severe burn
- Malignancy
- Neutropenia or pancytopenia.
- Severe chronic dis.

sig. • CIP : « Tense Vesicles or pustules that surrounded by narrow pink - violaceous

Hgic Pustule
Surr. by Erythema

↓ 12 hrs.

Necrotic gangrenous ulcer
surr. by Erythema

Halo » → Hgic → rupture →

round ulcers with necrotic black center

→ Site : perineal & gluteal > axillae > Extremities > Trunk

- Healthy infants may develop lesions in perineal area after antibiotic therapy in conjunctⁿ e maceratⁿ of diaper area.

• Diagnosis ① Clinically: → Classic Pustule → ulcer.

② Vesicle Aspirate & Culture → G-ve bacilli

(G-ve) (G-ve) (G-ve) ← ③ Blood Culture (if ass. Septicemia)

④ SKIN Biopsy → 2 Biopsies For Histopath. & Culture.

Treatment



① Early institution of IV antibiotics

Aminoglycosides + Antipseudomonal penicillin (Piperacillin).

+

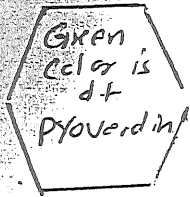
GMCSF (adjuvant H to ++ myeloid precursors in pts e Neutropenia).

② IF failed medical → Surgical debridement.

5. Green Nail Synd. & Green Foot Synd.

Green Nail: onycholysis + greenish discoloration
(in the separated areas)

(in chr. water users)



Gentamycin ± / Neomycin (✓)

Silver Sulfadiazine ±
effective / Mupirocin ±

١٤: ① Acetic acid 1% Soak حشيشه

② Trimming of the onycholytic areas + Neosporin Sol. (حشيشه)

Green Foot Synd: d.t. Colonization of rubber
Sports shoes with *P. auriginosa*

6. External Otitis:

• Common in Swimmers

• CIP: Erythema, Edema, Swelling & Pain.

• ١٤: ① prophylactic حشيشه: Otic Domiboro Sol.

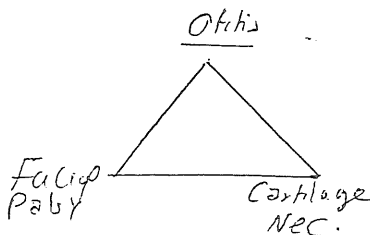
② Active ١٤: acetic acid + Cs Sol.

• DD:

١٤: ① CD: حشيشه
الموجود
... لا

① CD: حشيشه but
Dermatitis may extend down the
side of neck in pattern
suggesting drainage of suspension.

② Mg Otitis Externa: Severe Type affecting



Immuno Suppressed ch' B/ Severe
Erythema, Edema, Pain & Purulence &
± ass. e Facial palsy & Cartilage
Necrosis.

(١٤: Quinolones (4-6 wks))

③ Commercial Ear piercing of upper ear cartilage
± → Pseudom. e deformity.

Pyodermatitis / Pyoderma Vegetans

(PV)

PV. of Helwege

Blastomycosis like
Pyoderma... (Bact.)

Def. disorder ch by large Verrucous plaques & elevated

Borders & Multiple pustules.

Etiopath: ?? ... Bact. Inf + Immuno Suppression

Staph & Strep

Bact. & Fungal

Face & Flexures.

HIV
Leuk. Lymph.
U. Colitis
Alcoholism.

.. DO

(1) Vegetans d. - Pemphigus Veget.
- pemphigoid Veg.

Staph & E. coli

Immun. Supp.

granules
Similar
to Actinomyc.

(2) Botryomycosis: Bact. Inf. in Immuno.
Compromised Individual.

= التهاب بكتيري - التهاب فطري

Basophilic grs. Similar to that
of Actinomycosis.

(3) Deep fungal inf. - Phycomycosis
- Coccidioidomycosis.

(4) others: Brucella Iodo dermo, Mycobact.
Inf., Giant KA & PG.

(5) Pyodermatitis. Prostatitis Vegetans: as

Py. veget. but

affect mucous sites (oral &
Ass. @ U. Coliti, Flexure)

± am & IBD
cut PG
ulcerative

oral PG

→ (6) Pyostomatitis Vegetans

HP (2)

(i). Pseudoeplitheliom-
atous Hyperplasia

(ii) Epidermal
Abscesses

Staph is Isolated

TTT

(i). Antibiotics + local - Curvetage
Alum. subacetate
dressing

(ii) 20% human Albumin (100 ml) Infusion
Fr 5ds → 40mg, Cs Fr month.

(iii) Correction of Immuno Suppression.

Pyoderma Vegetans

(Chronic Botryomycosis & Actinomycosis)

Inf. Ch BY $\left\{ \begin{array}{l} \text{pustules} \\ \text{Verrucous plaques} \\ \text{ulcerated} \end{array} \right.$

Etiopath: Inf. + Immuno suppression

- Staph
- Staph
- Trichophyton
- Mentagrophytes
- HIV
- Ulcerative Colitis \rightarrow pyoderma
- Leukemia
- LE Nephritis
- Alcoholism & malnutrition

H-S may cause react (Tyk III) \rightarrow Pyod. Veget.

pt. is pyoderma + pyostomatitis + u. colitis has

Ant Bp 230 KD Antibodies

CIP: plaques of $\left\{ \begin{array}{l} \text{pustules} \\ \text{Verrucous} \\ \text{ulcerated} \end{array} \right.$ may affect any area even flexural area (DD from pemphigus veg.)

HP: Pseudoepitheliomatous Hyperplasia

+ Abscesses in $\left\{ \begin{array}{l} \text{dermis} \\ \text{Hyperplastic epid.} \end{array} \right.$

III: $\left\{ \begin{array}{l} \text{Etiology (Suppression)} \\ \text{Antibiotics: Ceftriaxone others Human Albumin + MPA} \\ \text{wound care: Curettage, Alum. Subacetate wash,} \\ \text{Zinc oxide, ILCs, disodium chromoglycate} \end{array} \right.$

Healing of ILCs pg edges
Leg. ulcers. II

may be related to Botryomycosis

(1) Pyoderma Vegetans

(2) it + Actinomycosis

Parula \rightarrow Bacteriosis

(3) Isolated entity

Etiopath: Trauma + Inf. + Immuno def.

Staph.
Bacteroides
E. coli

Fr long

inf. may affect: Skin, S.C.T, Ms, Bone, Int. organs

CIP 1. S.C nodules
2. large verrucous
3. ulcers

4. Granules \rightarrow pus is sulphur or yellow granules.

II. Surgical debridement

(Actinomycosis)

(S) Mycetozoa: granules
Suppurative inflammation
Center: Basophilic
Beclies (Bact debris)
periphery: Homogeneous eosinophilic
halo, 2 yr to 10 yr
PAS stain, Gram, Igs (Splendore phenom).